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Scotopic sensitivity during adulthood

Gregory R. Jackson, Cynthia Owsley *

Department of Ophthalmology, School of Medicine, University of Alabama at Birmingham, 700 S. 18th Street, Suite 609, Birmingham, AL 35294-0022, USA

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Abstract

Older adults typically exhibit about a half log unit loss in scotopic sensitivity that cannot be attributed to optical factors and retinal disease suggesting a neural origin. Little is understood about the developmental course of this neural deficit as to whether it first appears in late life or gradually emerges during the course of adulthood. To address this developmental issue, scotopic sensitivity was measured in 94 adults ranging in age from the 20s to the 80s. Thresholds were measured at 27 test loci within a 18° radius field. Analogous measurements were made for photopic sensitivity. Fundus photography and a grading scale were used to characterize macular health in subjects over age 49 in order to control for macular disease. Scotopic sensitivity decreased at a rate of 0.08 log units per decade; this decline was better fit by a single line model, not a bilinear model, implying that the impairment does not suddenly emerge in late life but gradually appears over the course of adulthood. Photopic sensitivity also decreased in a linear fashion at a rate of 0.04 log units per decade. Under these test conditions, the rate of scotopic sensitivity decline during adulthood was about double the rate of photopic sensitivity decline.

Keywords: Ageing; Scotopic sensitivity; Rods; Human

1. Introduction

Even when older adults are in good retinal health, they exhibit on average a 1 log unit elevation in absolute threshold under dark-adapted conditions (Gunkel & Gouras, 1963; Sturr, Zhang, Taub, Hannon & Jackowski, 1997; Jackson, Owsley, Cordle & Finley, 1998; Jackson, Owsley & McGwin, 1999). Scotopic sensitivity deficits may contribute to difficulty with visual activities performed under low luminance and at night (Kosnik, Winslow, Kline, Rasinski & Sekuler, 1988; Kline, Kline, Fozard, Kosnik, Schieber & Robert, 1992; Mangione, Berry, Spritzer, Janz, Klein, Owsley et al., 1998; Owsley, Stalvey, Wells & Sloane, 1999). Prior work (Sturr et al., 1997; Jackson et al., 1998, 1999) has indicated that even after pre-retinal factors (i.e. pupillary miosis and increased lens density) and criterion effects are taken into account, a half log unit of threshold elevation remains, suggesting that aging-related neural factors also contribute to sensitivity impairment in the elderly. A number of neural causes for this visual deficit have been suggested, including aging-related delays in rhodopsin regeneration (Liem, Keunen, van Norren & van de Kraats, 1991; Jackson et al., 1999), changes in the metabolic support structures of the retina (Domey, McFarland & Chadwick, 1960; Grindle & Marshall, 1978; Pauleikhoff, Harper, Marshall & Bird, 1990; Bird, 1992; Moore, Hussain & Marshall, 1995), rod loss (Gao & Hollyfield, 1992; Curcio, Millican, Allen & Kalina, 1993; Sturr et al., 1997), ganglion cell loss (Gao & Hollyfield, 1992; Curcio & Drucker, 1993; Sturr et al., 1997), and post-receptoral and cortical dysfunction in the neural pathway (e.g. Trick, Trick & Haywood, 1986; Porciatti, Burr, Morrone & Fiorentini, 1992; Muir, Barlow, & Morrison, 1996; Schefrin, Bieber, McLean & Werner, 1998; Schefrin, Tregear, Harvey & Werner, 1999).

Little is understood about the developmental course of older adults' neural deficit in scotopic sensitivity in terms of whether it first appears in later life or gradually emerges during adulthood. Two previous cross-sectional studies have examined scotopic sensitivity in

* Corresponding author. Tel.: +1-205-3258635; fax: +1-205-3258692.

E-mail address: owsley@eyes.uab.edu (C. Owsley).

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adults from their 20s to 60s, and also corrected for pre-retinal factors. Pulos (1989) reported that there were no differences in scotopic sensitivity in a sample of older adults ranging in age from 19 to 61 years old after lenticular density and pupil differences were taken into account. It appears that there was insufficient statistical power to detect age differences in this study since only 23 subjects spanning four decades were tested. Hammond, Wenzel, Rivera, King and Choate (1998a) measured scotopic sensitivity at a single test point presented at 6° eccentricity in subjects ranging in age from 20 to 63 years. Pre-retinal factors were taken into account. There was no change in scotopic sensitivity between the 20s and early 60s, however analyses on certain subgroups of the sample revealed aging-associated deficits (e.g. past smokers, those who never smoked). The authors point out that the aging effects were non-linear because sensitivity loss was only exhibited by the older adults and was largely due to subjects aged 45–65-years-old; however, the superiority of a bilinear or exponential fit was not statistically verified. A further problem with prior studies is that neither Hammond et al. (1998a) nor Pulos (1989) used standard and reliable criteria for defining normal retinal health. Instead, these studies used self-report (Hammond et al., 1998a) or clinical judgment (Pulos, 1989) to define subjects in good eye health. Both of these methods are subjective, even in the case of clinical judgment in which there are wide individual differences in what clinicians call normal health in the elderly. Thus it is difficult to compare their data to other studies. Furthermore, basing case definitions for normal aging versus disease on these subjective methods is problematic since age-related macular degeneration (AMD) is relatively common in older adults (Leibowitz, Krueger, Maunder, Milton, Kini, Kahn et al., 1980; Klein, Klein & Linton, 1992) and is known to impair scotopic sensitivity (Sunness, Massof, Johnson, Finkelstein & Fine, 1985; Owsley, Jackson, Cidiceyan, Huang, Fine, Ho et al., 2000). These methodological issues suggest that the question of scotopic sensitivity changes during adulthood deserves further study.

Also unresolved in the literature is whether the relative rates of light sensitivity decline in photopic versus scotopic vision during adulthood is the same or different. Photopic sensitivity for all three cone types is also reduced in older adults (Haegerstrom-Portnoy, Hewlett & Barr, 1988; Hammond, Wooten & Snodderly, 1998b; Johnson, Adams, Twelker & Quigg, 1988; Werner & Steele, 1988). Anatomical work has indicated that although the density of both rods and cones tend to decline during aging, this effect is dramatic for rods and minimal for cones (Curcio et al., 1993; Gao & Hollyfield, 1992). Furthermore, older persons indicate more serious visual difficulty with activities at night than during the day (Kosnik et al., 1988; Kline et al., 1992; Mangione et al., 1998). These findings lead to the hypothesis that scotopic sensitivity may exhibit a faster rate of decline during adulthood than does photopic sensitivity. No previous study has compared and contrasted the relative impairment rates for scotopic and photopic light sensitivity across adulthood in the same sample of clinically well-characterized observers.

This study addressed whether scotopic sensitivity undergoes changes during adulthood and in doing so overcomes methodologic limitations of the earlier work. First, a large sample was tested covering a wide range of ages from the 20s to the 80s, so statistical power was adequate. Older adults were over-sampled relative to younger adults because between subject variability typically increases with age in visual tasks (Owsley & Sloane, 1990). Second, 'normal eye health' in older adults was defined in terms of a standard grading scale of macular health, rather than by self-report or clinician judgment. Third, scotopic sensitivity was assessed at 27 points from the central 18 radius field involving both the horizontal and vertical meridian, an area where aging-associated rod loss is maximal (Curcio et al., 1993). This allowed us to assess whether there are regional variations in the presence or extent of scotopic deficits during aging. Previous studies focused on either a single test point (Hammond et al., 1998a) or in a single retinal region (Pulos, 1989). Finally, statistical techniques were utilized to determine whether any observed change in scotopic impairment as a function of decade is linear, or whether the magnitude of the deficit first emerges in the later decades of life. This study also afforded us the opportunity to measure photopic and scotopic light sensitivity in the same subjects in order to compare their relative rates of decline during adulthood.

2. Method

2.1. Subjects

The sample consisted of 94 adults ranging in age from the 20s to 80s (20s $n = 10$, 30s $n = 8$, 40s $n = 10$, 50s $n = 20$, 60s $n = 21$, 70s $n = 17$, 80s $n = 8$), with 55 females and 39 males, and 86 Caucasian, seven African American, and one Asian. Informed consent was obtained from all subjects after the nature of participation was explained. Subjects had 20/25 acuity or better (best-corrected, distance) in both eyes and were free of a diagnosis of cataract, AMD, glaucoma, diabetes, or any other eye or neurological condition known to compromise visual function, as indicated by the medical record from a comprehensive eye examination within 12 months of testing. To ensure that older subjects did not have AMD, those over age 49 underwent stereographic fundus photography on the eye to be tested. An

experienced grader used a standard scale of macular health to evaluate photographs, described previously (Jackson et al., 1998, 1999). The grader was unaware of the subject's vision status, prior ocular diagnoses, and age. Fundus grading indicated that no subjects over age 49 exhibited geographic atrophy or choroidal neovascularization (grades 3, 4, or 5). Thirty-one subjects had grade 0, 14 grade 1, and 21 grade 2.

2.2. Procedure

Photopic and scotopic sensitivity testing was performed with a modified Humphrey Field Analyzer 640 (HFA) (Humphrey Instruments, Inc.), a computer-automated perimeter for measurement of scotopic and photopic light sensitivity described in detail elsewhere (Jackson et al., 1998, 1999). For photopic testing, the wavelength of the target was 600 nm (Ealing # 35-3821, FWHM 10.1, Peak 50%), and target size was a Goldmann size V (1.7° of visual angle) circular test spot projected for 200 ms into a Ganzfeld bowl with a background luminance of 10 cd/m^2. Light sensitivity was measured at 27 locations in the central 18° of the visual field. Thirteen test points were located on the horizontal meridian (± 2, ± 4, ± 8, ± 10, -12, ± 18; ± 12°) and 14 on the vertical meridian (± 2, ± 4, ± 8, ± 10, ± 12, ± 18; ± 12°). The HFA s full threshold procedure (4-2 modified staircase threshold strategy) was used to estimate sensitivity. The HFA expresses sensitivity values as decibels (dB) of an attenuated 10 000 apostilb light source. During testing the subject's head was situated on a chin/forehead rest at a distance of 30 cm from the Ganzfeld bowl. The subject was instructed to fixate on a small, red fixation light located at the center of the bowl, and on each trial, the task was to push a response button when the target was detected. Targets were randomly presented at one of the 27 test locations. Fixation errors, false positives, and false negatives were recorded. If any of these error rates exceeded 10%, the subject's data was excluded from analysis. However, no subjects in the sample had to be excluded on this basis.

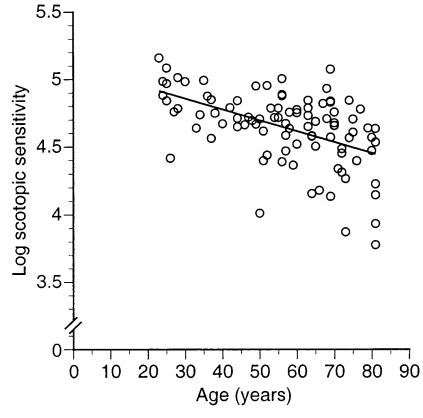

Fig. 1. A scatterplot of scotopic sensitivity as a function of age. Mean scotopic sensitivity decreased by 0.08 log units per decade.

For scotopic testing, subjects adapted to the dark for 40 min prior to beginning threshold measurement. Scotopic sensitivity was measured at the same test points as for photopic testing. The target was a 500-nm (Ealing # 35-3508, FWHM 7.4, Peak 50%), Goldmann size V (1.7° visual angle), circular test spot. The testing procedure was identical to that from photopic testing except that fixation was monitored manually by a infrared CCD camera. Our previous work with this apparatus indicated that test–retest reliability for this scotopic testing procedure is high ($r = 0.76$, $P < 0.002$) (Jackson et al., 1998).

Subjects were dilated with 1% tropicamide and 2.5% phenylephrine hydrochloride prior to testing. All subjects achieved a pupil diameter of ≥ 6 mm which was verified under scotopic conditions before and after testing. Subjects viewed the test target with their best optical correction for the test distance under these viewing conditions. The eye with better distance visual acuity was tested. Lens density of each subject's test eye was estimated psychophysically using an adaptation (Jackson et al., 1998, 1999) of a procedure previously described by Sample (Sample, Esterson, Weinreb & Boynton, 1988; Sample, Esterson & Weinreb, 1989) and Johnson (Johnson et al., 1988; Johnson, Adams & Lewis, 1989), which has high test-retest reliability (Sample et al., 1988). Prior to data analysis, each subject's scotopic sensitivity was corrected for his/her individual lens-density estimate using methods described previously (Jackson et al., 1998). Lens-density correction was not required for the cone-mediated thresholds because the lens' light absorption at 600 nm is insignificant (Wyszecki & Stiles, 1986).

3. Results

Subjects regardless of age group exhibited higher scotopic sensitivity at 8–12° eccentricity as compared with the most central area and more peripheral areas tested ($F[6,558] = 219.12$, $P < 0.0001$). Scotopic sensitivity averaged over the test field decreased at a rate of 0.1 log units per decade ($r = 0.58$, $P < 0.0001$), and after correcting for lens density it decreased at a rate of 0.08 log units per decade (see Fig. 1; $r = 0.50$, $P < 0.0001$). The aging-related deficit in scotopic sensitivity was diffuse across the test field in that it did not vary by eccentricity ($F[36,522] = 0.73$, $P = 0.88$) or by meridian ($F[6,87] = 0.77$, $P = 0.60$) within the 18° test field. To determine whether the rate of scotopic sensitivity decline (lens-corrected) increased in the later decades of life, a bilinear model (Owsley, Knoblauch & Katholi, 1992) was applied to the lens-corrected scotopic sensi-

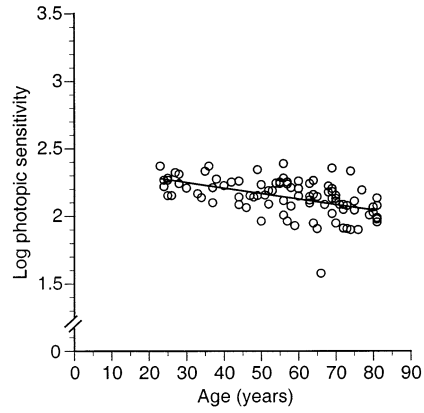

Fig. 2. A scatterplot of photopic sensitivity as a function of age. Mean photopic sensitivity decreased by 0.04 log units per decade.

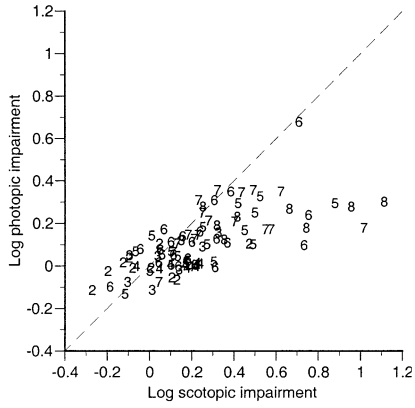

Fig. 3. For each subject, mean scotopic sensitivity impairment plotted as a function of mean photopic sensitivity impairment. Impairment for each individual was defined as the subject's average sensitivity across the test field subtracted from the average of adults in their twenties (see text for further details). The dashed diagonal line represents equal impairment in photopic and scotopic sensitivity under our test conditions. Numbers represent the age of each subject: 2 = 20s, 3 = 30s, 4 = 40s, 5 = 50s, 6 = 60, 7 = 70s, 8 = 80s.

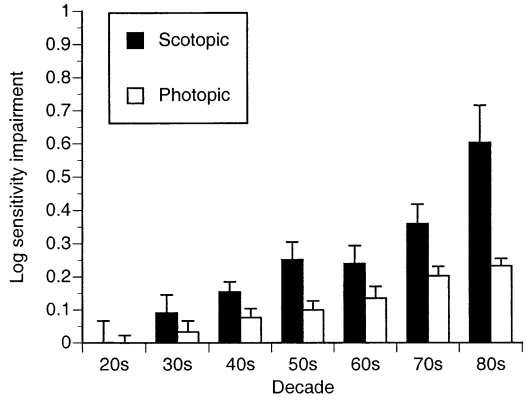

Fig. 4. Average scotopic and photopic sensitivity impairment plotted as a function of decade. Bars represent one standard error of the mean.

tivity as a function of age. The bilinear fit ($r^2 = 0.28$) was not superior to a simple linear fit ($r^2 = 0.25$) (χ^2 (2) = 4.03, $P = 0.13$), implying that that there was no increase in the rate of decline in later life in this sample of older adults in good retinal health. In addition, an exponential model ($r^2 = 0.10$) was not a significant improvement over the linear model ($r^2 = 0.25$) (χ^2 (1) = 0.01, $P = 0.92$); in fact the linear model accounted for more variance.

A parallel analysis was performed on the photopic data. Subjects regardless of age exhibited better photopic sensitivity in more central areas of the 18° test field than in the peripheral areas (F[6,558] = 340.88, $P < 0.0001$). Photopic sensitivity averaged across all 27 points in the test field decreased at a rate of 0.04 log units per decade as shown in Fig. 2 ($r = 0.52$, $P < 0.0001$). The aging-related deficit of photopic sensitivity was diffuse across the test field in that it did not vary by eccentricity (F[36,522] = 0.73, $P = 0.88$) or by meridian (F[6,87] = 0.33, $P = 0.92$) within the 18° test field. To determine whether the rate of photopic sensitivity decline increased in the later decades of life, a bilinear model was applied to photopic sensitivity as a function of age. However, the bilinear fit was not a significant improvement over a simple linear fit (χ^2 (2) = 0.97, $P = 0.62$).

Our older adult sample (> 49-years-old) contained a few older adults whose macula displayed one or more drusen ≥ 65 μm in diameter and/or focal hyperpigmentation (grade 2; see Jackson et al., 1998, 1999). In a further analysis, we used a more conservative definition of normal retinal health in the older subjects and restricted the older adults in the sample to only those with a fundus grade of 0 and 1 and eliminated those with grade 2. In this situation, scotopic sensitivity averaged across the test field decreased at a rate of 0.09 log units per decade ($r = 0.60$, $P < 0.00001$). The rate of decline in scotopic sensitivity when including (0.08 log unit per decade) versus omitting the grade 2s (0.09 log unit per decade) was not significantly different (F[1] = 0.00, $P = 0.97$). Similarly, the rate of photopic sensitivity decline with the older adults with grade 2 omitted from the sample (0.04 log units per decade) was not different from the rate of decline when they are included (0.04 units per decade) (F[1] = 0.01, $P = 0.93$).

For the purposes of Fig. 3, impairment in scotopic sensitivity for each individual was defined as the subject's average sensitivity across the test field subtracted from the average of adults in their twenties. Impairment in photopic sensitivity was defined for each individual subject in an analogous fashion. Fig. 3 displays each subject's scotopic impairment plotted against photopic impairment. By the middle to later decades of life, scotopic impairment is typically more severe than photopic impairment, as evidenced by the majority of points from older subjects lying below the diagonal in Fig. 3. Fig. 4 displays, averaged across subjects, the

magnitude of impairment under both photopic and scotopic conditions as a function of decade. Scotopic sensitivity impairment increased at a greater rate than photopic sensitivity impairment as a function of decade ($F[6,87] = 3.46,\ P < 0.0042$).

4. Discussion

With increasing age during adulthood from the 20s to 80s, adults exhibited decreases in scotopic sensitivity in the central 18° field that cannot be attributed to optical factors or retinal disease, implying an aging-associated neural origin for the impairment. This impairment does not suddenly emerge in later adulthood but instead gradually reveals itself over the course of adulthood. This is evidenced by the bilinear model not being superior to the single line model relating age and scotopic sensitivity. In addition, scotopic sensitivity decreases during adulthood at about twice the rate of photopic sensitivity decline. This phenomenon is also consistent with earlier findings that acuity and spatial contrast sensitivity impairments in older adults are accentuated at low luminance (Adams, Wang, Wong & Gould, 1988; Sloane, Owsley & Alvarez, 1988; Sloane, Owsley, & Jackson, 1988). It is possible that cone-mediated dysfunction would be more severe under different stimuli and background conditions, which could effectively accelerate the rate of decline in cone-mediated function during adulthood. For example, cone dysfunction in early AMD, a common disease in the elderly, is accentuated under low luminance conditions and is less obvious under high luminance conditions (Sunness, Rubin, Applegate, Bressler, Marsh, Haekins et al., 1997). Thus the severity of cone-mediated sensitivity impairment in older adults may be affected by background light level and possible rod intrusion. However, it is fair to conclude on the basis of the present data that light sensitivity measured under dark-adapted conditions declines at a more rapid rate during adulthood than does light sensitivity measured under photopic conditions.

What are the possible neural explanations for the aging-related loss in scotopic sensitivity? As discussed earlier, Curcio et al. (1993) found that during adulthood, rod density decreased in the peri-foveal region of retina, whereas cone density was relatively unaffected. Previously, we examined the spatial distribution of aging-related scotopic sensitivity loss to determine if we could identify a psychophysical correlate to these anatomical findings (Jackson et al., 1998). We did not find heightened scotopic sensitivity loss in the perifovea where maximal rod loss occurs compared with the periphery where rod loss is minimal. As verified in the present study, the magnitude of scotopic sensitivity deficit appeared spatially uniform at all test locations within the peri-fovea. Curcio et al. (1993) point out that it is possible that rods 'compensate' for rod death by filling in the gaps left by degenerating rods, which could maintain the quantum catching ability of the retina despite rod loss. Thus, it is possible that this aging-related rod loss actually has little impact on absolute threshold, leading one to search elsewhere for explanations for older adults' scotopic sensitivity deficit.

Another possible source for the scotopic sensitivity impairment is an alteration of the retinal pigment epithelium and Bruch's membrane that impedes the passage of vitamin A or other nutrients to the rod photoreceptors (Curcio, Owsley & Jackson, in press). A localized scarcity of vitamin A would slow the visual cycle, the biochemical pathway responsible for rhodopsin regeneration. This slowing in rhodopsin regeneration could present itself as scotopic sensitivity loss if absolute threshold is measured before complete adaptation to darkness has occurred. To address this issue, we compared the rate of rod-mediated dark adaptation of these same 94 subjects as measured in an earlier study (Jackson et al., 1999) to their scotopic sensitivity, as measured in this study. The rate of rod-mediated dark adaptation was slowed with increasing age. However, the rate of rod-mediated dark adaptation was not correlated with scotopic sensitivity ($r(94) = 0.09,\ P = 0.40$). That is, those older adults with the most severe delays in dark adaptation did not have the most severe deficits in scotopic sensitivity. Thus, while it may be reasonable to consider visual cycle perturbations as an important mechanism underlying older adults' delay in dark adaptation, it appears that one must look elsewhere to fully explain their steady-state scotopic sensitivity deficit at a zero background. Another potential candidate mechanism is that during the aging process there may be changes in gain control in the rod itself, in other neurons in the retina, and/or in cortex that could cause scotopic threshold elevations. These possibilities deserve further examination especially in light of recent findings that ganglion cell density decreases during aging (Gao & Hollyfield, 1992; Curcio & Drucker, 1993) and that post-receptoral visual function changes in late adulthood (Trick et al., 1986; Porciatti et al., 1992; Muir et al., 1996; Schefrin et al., 1998, 1999).

The progressive loss of light sensitivity in darkness during adulthood may help explain greater visual difficulties at night and under low illumination as we age. Further work should explore the relationship between these psychophysical deficits in scotopic vision and both self-reported and performance deficits at night and under dim illumination. In addition, the age-associated trends emerging from these cross-sectional data await longitudinal verification.

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